7.0 RISK CHARACTERIZATION

Risk characterization is performed to describe and estimate the magnitude of risk and hazard from emissions from combustion units at TOCDF and CAMDS. Risk characterization involves the calculation of the upper-bound excess lifetime cancer risks (risk) and noncarcinogenic hazards (hazard) for each pathway and receptor evaluated in the risk assessment (U.S. EPA 1998a). The risks and hazards for each pathway will then be summed for specific receptors—across all exposure pathways—to obtain an estimate of total individual risk and hazard for each receptor.

The probability of carcinogenic and noncarcinogenic effects occurring in exposed populations will be evaluated using the exposure parameters, models, and toxicity values proposed in this protocol. Carcinogenic risk is defined as the probability that a receptor will develop cancer, based on exposure and toxicity assumptions. The CSF or URF is used to estimate the upper bound lifetime probability that an

individual will develop cancer as a result of exposure to a particular level of a potential carcinogen. A noncarcinogenic hazard is the potential for developing noncarcinogenic health effects as a result of exposure to COPCs, averaged over an exposure period. A hazard is a ratio of the magnitude of a receptor's potential exposure to a standard exposure level (RfD or RfC). The standard exposure level is calculated over an exposure period similar to the receptors and is not expected to pose a threat of a likely adverse health effect to potential receptors. Section 7.1 presents the methods for calculating cancer risk, and Section 7.2 presents the methods for calculating noncancer hazards. Section 7.3 discusses evaluation of the breast milk pathway. Section 7.4 discusses the evaluation of acute exposure from direct inhalation. Lastly, Section 7.5 describes the target levels.

7.1 METHODS FOR CALCULATING CANCER RISK

Carcinogenic risk will be estimated using the following formula:

$$CancerRisk = LADD * CSF \qquad \text{Equation 7-1}$$

where:

LADD = Lifetime average daily dose (mg/kg-day)
CSF = Cancer slope factor $(mg/kg\text{-}day)^{-1}$

A receptor may be exposed to more than one COPC within the same pathway. To evaluate pathway-specific cumulative risk, the total risk for all COPCs is evaluated using the following formula:

$$Cancer\ Risk_T = {}_i Cancer\ \ Risk_i \qquad \text{Equation 7-2}$$

where:

$Cancer\ Risk_T$ = Total cancer risk for a specific exposure pathway
$Cancer\ Risk_i$ = Cancer risk for COPC *i* for a specific pathway

For evaluating multiple combustion units and through multiple pathways, risk will be summed across the receptor-exposure pathway combinations and then summed for all combustion sources. The total risk posed to a receptor is the sum of total risks from each individual exposure pathways, and is calculated with the following formula:

$$Total\ Cancer\ Risk = Cancer\ Risk_T \qquad \text{Equation 7-3}$$

where:

Total Cancer Risk	=	Total cancer risk from multiple exposure pathways
Cancer $Risk_T$	=	Total cancer risk for a specific exposure pathway

The equations used to calculate dose and risk levels are presented in Appendix E. Appendix C presents the oral CSFs and inhalation URFs for the COPCs for TOCDF and CAMDS.

7.2 METHODS FOR CALCULATING NONCANCER HAZARDS

U.S. EPA (1989; 1998a) risk assessment methods assume that noncarcinogenic effects exhibit a threshold or a level of exposure below which no adverse effects will be observed (U.S. EPA 1998a). The potential for noncarcinogenic health effects will be evaluated in two ways (U.S. EPA 1998a): (1) by comparing an exposure estimate to an RfD for oral exposures and (2) by comparing an estimated chemical-specific air concentration to the RfC for direct inhalation exposures. Comparing exposure estimates and COPC-specific air concentrations to RfDs and RfCs results in the hazard quotient (HQ), which will be calculated as follows:

$$HQ = \frac{ADD}{RfD}\left(for\ oral\ or\ dermal\ exposures\right) \qquad \text{Equation 7-4}$$

$$HQ = \frac{C_a}{RfC}\left(for\ inhalation\ exposures\right)$$

where:

HQ	=	Hazard quotient (unitless)
ADD	=	Average daily dose (mg/kg-day)
C_a	=	Total COPC air concentration (mg/m^3)
RfD	=	Reference dose (mg/kg-day)
RfC	=	Reference concentration (mg/m^3)

A receptor can be exposed to multiple COPCs causing noncarcinogenic health effects. The hazard index (HI) is the total noncarcinogenic hazard attributable to exposure to all COPCs through a single exposure pathway. The HI will be calculated as follows:

$$HI = \sum_i HQi \qquad \text{Equation 7-5}$$

where:

HI	=	Total hazard for a specific exposure pathway
HQ_i	=	Hazard quotient for COPC *i*

As discussed before, a receptor may be exposed to COPCs associated with noncarcinogenic health effects through more than one exposure pathway and from more than one source. A receptor's total hazard is the sum of the HIs for each of the exposure pathways. This is expressed in the following calculation:

$$Total\ HI = \sum HI \qquad \text{Equation 7-6}$$

where:

Total HI	=	Total hazard from multiple exposure pathways
HI	=	Total hazard for a specific exposure pathway

Further evaluation is necessary if a total HI for an exposure pathway exceeds the target level (U.S. EPA 1998a). This might occur because (1) one or more COPCs with an HQ exceeds the target hazard level or (2) the sum of several COPC-specific HQs, each less than the target hazard level, is greater than one. If at least one COPC-specific hazard is greater than the target level, it will be interpreted as an indication of potential noncarcinogenic health effects. However, if the summation of several COPC-specific HQs (all less than the target level) is greater than one, COPC-specific hazards will be summed according to the major health effects and target organs and systems (U.S. EPA 1998a). The equations used to calculate the dose and hazard are presented in Appendix F. Appendix D presents the oral RfDs and inhalation RfCs for the COPCs for TOCDF and CAMDS

7.3 METHODS FOR EVALUATING THE BREAST MILK PATHWAY

The breast milk pathway is evaluated to assess the potential risk to nursing infants exposed to PCDDs and PCDFs ("dioxins"). This pathway is evaluated because of concern about the potential for infants to be exposed to these substances and their sensitivity to them (U.S. EPA 1996a). While other COPCs potentially found in emissions may exhibit the same characteristics as dioxins, the data for evaluating the risk to infants is insufficient. Exposure to dioxins is of concern because these substances readily accumulate in lipids and have been detected in breast milk. Infants are of particular concern because, on a body weight basis, they are potentially exposed to higher doses than adults while breast feeding. In addition, infants are susceptible to adverse developmental effects caused by these substances.

The breast milk pathway for dioxins will be evaluated in the HHRA for the residential and subsistence rancher exposure scenarios. The method recommended by U.S. EPA (1998a) will be used to quantify risks from infant exposure to mother's breast milk. The methodology, however, has some limitations and is currently under review by U.S. EPA. In the dioxin reassessment, U.S. EPA is currently considering whether the average daily dose is the most appropriate metric to evaluate exposure (DSHW 2000c). Section 7.3.1 presents the methodology recommended in U.S. EPA (1998a) and Section 7.3.2 provides a summary of the limitations and sources of uncertainty associated with the breast milk pathway.

7.3.1 U.S. EPA-Recommended Methodology

U.S. EPA (1998a) recommends comparing estimated dioxin exposures from emissions with national average background exposure levels (60 picograms (pg) TEQ/kg BW-day for nursing infants). If exposures to emissions during the exposure duration of concern are low compared to 60 pg/kg BW-day for infant exposures, then U.S. EPA (1998a) assumes that these emissions do not cause noncancer effects. In some cases, noncancer effects may be significant to infants even when dioxin emissions are lower than the national average background exposure level. However, this comparison to national average background levels was determined based on several policy considerations made by U.S. EPA. This methodology is currently recommended by U.S. EPA and will be implemented in the HHRA.

Two steps are used to estimate infant exposure to dioxins through breast milk. First, the concentration of dioxins in milk fat of breast milk will be calculated:

$$C_{Milkfat} = \frac{m \bullet 1\,x\,10^{9} \bullet h \bullet f_1}{0.693 \bullet f_2} \qquad \text{Equation 7-7}$$

where:

$C_{Milkfat}$	=	Concentration of dioxin in milk fat of breast milk for a specific exposure scenario (pg COPC/kg milk fat).
M	=	Average maternal intake of dioxin for each adult exposure scenario (mg COPC/kg BW-day)
$1\,x\,10^{9}$	=	Units conversion factor (pg/mg)
h	=	Half-life of dioxin in adults (days)
f_1	=	Fraction of ingested dioxin that is stored in fat
f_2	=	Fraction of mother's weight that is fat

Then, the average daily dose of dioxins for an infant exposed to contaminated breast milk will be calculated:

$$ADD_{infant} = \frac{C_{milkfat} \bullet f_3 \bullet f_4 \bullet IR_{milk} \bullet ED}{BW_{\text{inf } ant} \bullet AT} \qquad \text{Equation 7-8}$$

where:

ADD_{infant}	=	Average daily dose for infant exposed to contaminated breast milk (pg COPC /kg BW-day)
$C_{milkfat}$	=	Concentration of COPC in milk fat of breast milk for a specific exposure scenario (pg COPC /kg milk fat)
f_3	=	Fraction of mother's breast milk that is fat (unitless)
f_4	=	Fraction of ingested COPC that is absorbed (unitless)
IR_{milk}	=	Ingestion rate of breast milk by the infant (kg/day)
ED	=	Exposure duration (year)
BW_{infant}	=	Body weight of infant (kg)
AT	=	Averaging time (year)

The estimated dioxin daily dose to nursing infants will then be compared to the U.S. EPA-recommended background concentration of 60 picograms per kilogram per day (pg/kg/day). The equations and parameter values that will be used to evaluate adult exposure to dioxins, and infant exposure to dioxins in breast milk, are presented in Appendix E.

7.3.2 Limitations

The quantification of risks from infant exposure to dioxins from mother's milk involves some major limitations that may potentially affect the results or their interpretation. The following are some of the data limitations associated with the breast milk pathway (U.S. EPA 1997):

- Data are not available for infants under 1 month of age
- Data are not presented on a body weight basis. Body weight data may significantly affect infant sensitivity and exposure to dioxins.
- Subpopulations of mothers that breast feed longer than 1 year are not represented in the available studies

There is also a mass balance problem related to the calculated infant uptake of dioxins exceeding the total maternal dose received (Kerger and others 1999). Several assumptions tend to overestimate the dose or body burden of the breast-fed infant. The following assumptions may potentially overestimate the infant uptake of dioxins:

- Maternal body burden accumulated over 30 years of daily exposure is used to calculate infant dose via breast milk fat. Daily exposure over 30 years overestimates maternal exposures to dioxins.
- There is a lack of consideration of infant body burden versus time. Infant exposures and sensitivity will decrease as a function of time.
- The excretion half-life of dioxins is the same in both mother and infant. These should be derived for the mother and infant separately, which will lower infant uptake of dioxins.

If the 60 pg/kg/day target level for the breast milk pathway is exceeded, the limitations of the U.S. EPA methodology will be discussed as an uncertainty in the HHRA.

7.4 METHODS FOR EVALUATING ACUTE EXPOSURE FROM DIRECT INHALATION

In addition to long-term chronic effects, short-term or acute effects from direct inhalation of vapor phase and particle phase COPCs will be evaluated. It is assumed that short-term emissions will not have a significant impact through the indirect exposure pathways (as compared to impacts from long-term emissions). Therefore, acute effects will only be evaluated through the short-term (maximum 1-hour) inhalation of vapors and particulates exposure pathway of the acute risk scenario. In order to establish

acute inhalation exposure criteria (AIEC), it will be necessary to identify and evaluate (1) existing guidelines for acute inhalation exposure, and (2) existing hierarchal approaches for developing acute inhalation exposure levels. Hierarchal approaches are composed of existing guidelines for acute inhalation exposure, ranked in order of applicability and technical basis, and all being protective of the general public. It should be noted that hierarchical approaches are needed within this approach because no single organization or methodology has developed acute criteria values or benchmarks for all of the COPCs.

Acute inhalation exposure guidelines and criteria are (1) designed to protect a variety of exposure groups, including occupational workers, military personnel, and the general public; (2) based on varying exposure durations up to 24 hours in length; and (3) intended to protect against a variety of toxicity endpoints ranging from discomfort or mild adverse health effects to serious, debilitating, and potentially life-threatening effects, up to and including death.

U.S. EPA (1998a) recommends the following approach based on existing acute inhalation values that do not require the use of arbitrary safety factors and are intended to protect the general public from discomfort or mild adverse health effects over 1-hour exposure periods. It includes level 1 acute inhalation exposure guidelines (AEGL-1), level 1 emergency response planning guidelines (ERPG-1), and level 1 acute toxicity exposure levels (ATEL-1), supplemented with Department of Energy's (DoE) temporary emergency exposure limits (TEEL) and the Subcommittee on Consequence Assessment and Protective Actions (SCAPA) toxicity-based approach. The hierarchal approach is summarized below:

1. AEGL-1
2. ERPG-1
3. TEL-1
4. Level one-TEEL (TEEL-1)
5. SCAPA toxicity-based approach

This preference is based on the (1) applicability to a 1-hour exposure duration for protection of the general public (versus only occupational exposure), and (2) level of documentation and associated review.

To obtain a COPC-specific AIEC, the AEGL-1 values specific to the COPC of interest will be reviewed. AEGL-1 values are currently available for 12 compounds. If there is not an available AEGL-1 value for a respective COPC, the ERPG-1 values will be reviewed until an AIEC value is obtained for the COPC of interest.

To characterize the potential for adverse health effects from acute exposure to COPC-specific emissions, the acute air concentration resulting from maximum emissions over a 1-hour period will be compared to the COPC-specific AIEC to calculate the acute HQ as follows:

$$AHQ_{inh} = \frac{C_{acute} * 0.001}{AIEC} \qquad \text{Equation 7-9}$$

where:

AHQ_{inh}	=	Acute hazard quotient (unitless)
C_{acute}	=	Acute air concentration (:g/m^3)
AIEC	=	Acute inhalation exposure criteria (mg/m^3)
0.001	=	Conversion factor (mg/:g)

Acute HQs will be calculated at the selected acute exposure scenario locations for COPCs specific to emissions from each unit and from all units combined. Target levels for acute HQ evaluation is a risk management decision and will be set by DSHW.

7.5 TARGET LEVELS

The target levels for the risk assessment are determined by DSHW (the permitting authority). If the calculated values for the endpoints are equal to or less than the target levels, no additional evaluation is required. If the calculated values for the endpoints are greater than the target levels, additional analysis or mitigation is warranted. The risk assessment target levels are:

Endpoint	**Target Levels**
Carcinogenic effects	1×10^{-5}
Noncarcinogenic effects	0.25
Acute Inhalation Exposure	To be determined
Adult–Noncarcinogenic dioxin exposure	1 pg/kg-day
Nursing Infant–Noncarcinogenic dioxin exposure	60 pg/kg-day

The target level for carcinogenic effects of 1×10^{-5}, or 1 in 100,000, is the same target level used for the screening risk assessment (ATK 1996). The 1×10^{-5} is within the range outlined in the National Contingency Plan and is consistent with existing DSHW rules and policies. The 1×10^{-5} value is interpreted to mean that at the calculated exposure, a person's chance of getting cancer as a result of that exposure is no higher than 1 in 100,000. Another way of interpreting 10^{-5} is that if 100,000 people were all exposed to a chemical at the same levels, a maximum of one excess cancer would occur. Note that the cancer target levels are interpreted as upper bounds; the actual number of cancers would likely be less and could be zero. As a point of comparison, the lifetime cancer rate in the United States is 1 in 2 for males and 1 in 3 for females (American Cancer Society 1996).

The target level for noncarcinogenic effects of 0.25 (hazard index, see Section 7.2) is the same target level used for the Screening Risk Assessment (ATK 1996). Although no adverse health effects are predicted if the HI is less than one, the four-times-more-protective target level HI of 0.25 is selected as a method to account for potential, existing exposures from sources other than those at DCD.

The target level for acute inhalation exposures is a HI of one. No adverse health effects are predicted if the HQ is one or less. The potential for existing acute inhalation exposures from sources other than those at DCD is judged to be small for the 1-hour exposure time.

The target level for evaluating noncarcinogenic PCDD and PCDF exposures is 10 percent of the average dose attributable to background exposures in the United States. An HQ could not be calculated because of the lack of consensus on a safe dose (i.e., an RfD) for dioxin-like chemicals. The U.S. EPA (1998a) recommended approach is to compare the potential dose attributable to DCD emissions to the dose attributable to background. An additional 10 percent exposure added to existing (background) exposures is judged unlikely to result in any additional potential for adverse effects.

If the calculated values for carcinogenic and noncarcinogenic endpoints are less than the target levels, the conclusion is that potential exposures to emissions are safe. A calculated endpoint greater than the target level does not indicate an unsafe action or an unacceptable risk but does indicate that additional evaluation or mitigation is warranted.

The additional evaluation will focus on the COPCs and exposure pathways whose endpoints exceed the target levels. Many of the parameters and assumptions in the risk assessment are anticipated to

overestimate actual exposures. These parameters and assumptions can potentially be further refined based on site-specific conditions. If the endpoint for a potential future exposure pathway exceeds a target level, the conclusion of the additional evaluation may be to monitor for the completion of the pathway or to implement an environmental monitoring program. If the target levels are exceeded, mitigation options include modifying the operating conditions of the incinerators (e.g., feed rates, combustion conditions) or installing pollution control devices.